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Effect of Heating Rate on Evaporative Heat Loss in the Microwave-Exposed Mouse

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16. ABSTRACT

Male CBA/J mice were administered heat loads of 0-28 $J.g^{-1}$ at specific absorption rates (SARs) of either 47 or 93 $W.kg^{-1}$ by exposure to 2,450-MHz microwave radiation at an ambient temperature of 30 C while evaporative heat loss (EHL) was continuously monitored with dew-point hygrometry. At an SAR of 47 $W.kg^{-1}$ a threshold heat load of 10.5 $J.g^{-1}$ had to be exceeded before EHL increased. An approximate doubling of SAR to 93 $W.kg^{-1}$ reduced the threshold to 5.2 $J.g^{-1}$. Above threshold the slopes of the regression lines were 1.15 and 0.929 for the low and high SAR groups, respectively. Thus the difference in threshold and not slope attributes to the significant increase in EHL when mice are sxposed at a high SAR ($P < 0.02$). In separate experiments a SAR of 47 $W.kg^{-1}$ raised the deep body temperature of anesthetized mice at a rate of 0.026 C. s^{-1}, whereas 93 W.kg-1 raised temperature at 0.049 C.s-1. Hence the sensitivity of the EHL mode of heat dissipation is directly proportional to the rate of heat absorption and to the rate of rise in body temperature. These data contradict the notion that mammals have control over whole-body heat exchange only (i.e. thermoregulation) but instead indicate that the EHL system is highly responsive to the rate of heat absorption (i.e. temperature regulation).

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JOURNAL ARTICLE

Effect of heating rate on evaporative heat loss in the microwave-exposed mouse

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GORDON, CHRISTOPHER J. *Effect of heating rate on evaporative heat loss in the microwave-exposed mouse.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 53(2): 316–323, 1982.—Male CBA/J mice were administered heat loads of 0–28 $J \cdot g^{-1}$ at specific absorption rates (SARs) of either 47 or 93 $W \cdot kg^{-1}$ by exposure to 2,450-MHz microwave radiation at an ambient temperature of 30°C while evaporative heat loss (EHL) was continuously monitored with dew-point hygrometry. At an SAR of 47 $W \cdot kg^{-1}$ a threshold heat load of 10.5 $J \cdot g^{-1}$ had to be exceeded before EHL increased. An approximate doubling of SAR to 93 $W \cdot kg^{-1}$ reduced the threshold to 5.2 $J \cdot g^{-1}$. Above threshold the slopes of the regression lines were 1.15 and 0.929 for the low- and high-SAR groups, respectively. Thus the difference in threshold and not slope attributes to the significant increase in EHL when mice are exposed at a high SAR ($P < 0.02$). In separate experiments a SAR of 47 $W \cdot kg^{-1}$ raised the deep body temperature of anesthetized mice at a rate of 0.026°C · s^{-1}, whereas 93 $W \cdot kg^{-1}$ raised temperature at 0.049°C · s^{-1}. Hence the sensitivity of the EHL mode of heat dissipation is directly proportional to the rate of heat absorption and to the rate of rise in body temperature. These data contradict the notion that mammals have control over whole-body heat exchange only (i.e., thermoregulation) but instead indicate that the EHL system is highly responsive to the rate of heat absorption (i.e., temperature regulation).

open-loop gain; radiofrequency radiation

IN A GLOSSARY OF TERMS for thermal physiology published in 1973, Bligh and Johnson (4) defined temperature regulation and thermoregulation as synonymous. However, other investigators have attempted to distinguish these terms when describing the control of body temperature. For example, Snellen (21) asserted that temperature homeostasis in humans is a process of heat regulation rather than temperature regulation. Webb et al. (26) have shown in exercising humans that temperature per se is not the regulated parameter but is rather the end result of the control of heat loss. In a model of thermoregulatory control in humans, Houdas et al. (16–18) concluded that the system directly exerts control over the level of heat storage and indirectly has control over body temperature.

The terms thermoregulation and temperature regulation are etymologically different. The former implies the regulation of heat content, whereas the latter refers to the regulation of temperature in specific sites such as the rectum, skin, and hypothalamus. What is not understood, however, is whether the sensory-motor components of body-temperature control are equally as sensitive to total heat exchange as they are to the rate of heat exchange. In other words, does the system sense and respond similarly to heat stimuli of varying absorption rates (i.e., $J \cdot g^{-1} \cdot s^{-1}$) but of equal energy (i.e., $J \cdot g^{-1}$)? If the mammal is a thermoregulator then the overall motor response to a heat load should closely correlate with the absorbed heat load and be independent of the absorption rate. If, on the other hand, the mammal is a temperature regulator, then the magnitude of the motor response should be proportional to the rate of heat absorption.

The purpose of this study is to discern whether the mouse thermoregulates or temperature regulates when exposed to heat loads of varying intensity. As will be explained in MATERIALS AND METHODS, the use of a microwave-exposure system is ideal for this type of study because it allows for the administration of exact quantities of heat at varying rates of absorption. In this investigation evaporative heat loss is measured in mice exposed to 2,450-MHz microwave radiation at varying intensities and heat loads.

MATERIALS AND METHODS

The animals used in this study were 5-mo-old male CBA/J mice with a mean body weight of 34.6 ± 0.4 (SE) g. The mice were maintained at 22°C, 40–50% relative humidity, and a 12:12 LD photoperiod. The animals were previously acclimated to the microwave facility with periodic exposures approximately twice per week.

Microwave exposure. The animals were subjected to the absorption of whole-body heat loads by exposure to 2,450-MHz electromagnetic radiation inside a rectangular waveguide (5.8 × 11.2 × 30.5 cm). The schematic in Fig. 1 shows the path of the microwave radiation through the exposure system. When the microwaves pass through the waveguide there is a small but constant power loss due to leakage and attenuation in the cables and electrical connections and absorption by the Plexiglas animal holder (total loss 16%). When a mouse (or any biological material) is placed in the microwave field, a relatively large drop in power will be detected on the output side of the waveguide (i.e., P_{tran} in Fig. 1). By measuring the incident of the radiation entering (P_{inc}) and leaving the waveguide (P_{tran}) and the reflected power (P_{ref}) that emits from the mouse back to the microwave generator, the amount of power absorbed by the mouse can be calculated ($P_{abs} = P_{inc} - P_{ref} - P_{tran}$). A mouse will usually

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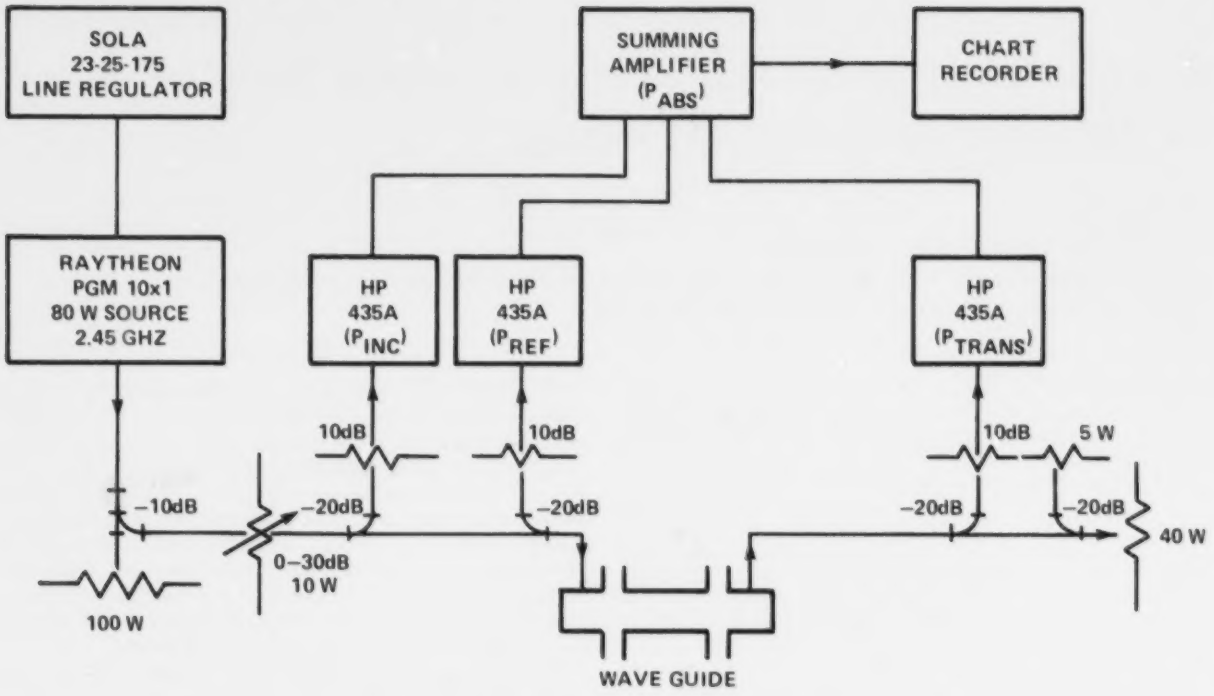

FIG. 1. Schematic of path of microwaves through waveguide-exposure system. 90% of power from generator is routed to a 100-W termination load while remaining power is passed through a 0- to 30-dB variable attenuator and then into waveguide. Hewlett-Packard 453A power meters are used to monitor incident (P_{inc}), reflected (P_{ref}), and transmitted power (P_{trans}) emitting to and from waveguide. A chart recorder continually monitors absorbed power, which is derived by integrating analog voltages from meters. Waveguide-exposure system is modified from that of Ho and Edwards (15).

absorb between 50 and 80% of the total incident power with the remainder passing into the terminal absorber. Absorbed power is continuously recorded by summing the analog voltage outputs from the power meters depicted in Fig. 1.

An example of the time course of absorbed power in a mouse during microwave exposure is shown in Fig. 2. The fluctuations in absorbed power are a result of the mouse changing position in the waveguide during exposure. Power absorption is maximal when the mouse is oriented longitudinally with respect to the long axis of the waveguide. Integrating the area under the curve in Fig. 2 (i.e., power, $W \cdot kg^{-1}$, over time, s) yields the total heat absorbed by the animal from microwave exposure. In the example of Fig. 2 the specific absorption rate (SAR) is equal to 97.2 $W \cdot kg^{-1}$, and the exposure time is 120 s, yielding a total heat absorption equal to 11.6 $J \cdot g^{-1}$.

The microwave-exposure system provides a way to raise the deep body temperature at a given rate and simultaneously calculate the amount of heat absorbed by the animal. For example, consider two exposure regimes, one with an SAR of 100 $W \cdot kg^{-1}$ over a period of 60 s and another at 50 $W \cdot kg^{-1}$ over 120 s. Both exposures result in identical heat loads to the animal (6,000 $J \cdot kg^{-1}$); however, a power level of 100 $W \cdot kg^{-1}$ will raise body temperature at approximately twice the rate of that at 50 $W \cdot kg^{-1}$. Hence the microwave-exposure system permits an opportune means of applying measurable heat loads of various absorption rates of unrestrained animals.

Since the mice were exposed at a thermoneutral ambient temperature of 30°C, the increase in body temperature during microwave exposure would, by elevating metabolism, produce an additional heat load. SARs of 47 and 93 $W \cdot kg^{-1}$ used in this study represent heat loads of approximately 6 and 12 times the resting metabolic heat production of the mouse. Hence the additional heat load accrued from the hyperthermic effect of microwave exposure represents a very small percentage of the total heat load administered.

Experimental apparatus. The body of the waveguide, excluding the electrical connections, was immersed in a water bath maintained at 30 ± 0.5°C. Two vents in the waveguide allowed for the continuous passage of air, previously dried over anhydrous calcium sulfate to a partial pressure of approximately 0.2 Torr. The air was drawn into the waveguide at a rate of 1 l/min and then, via copper tubing, into a General Eastern dew-point hygrometer sensor. Dew-point temperature was first converted to evaporative water loss (EWL) (27) and then to evaporative heat loss (EHL) by assuming that 1.0 g of evaporated water was equivalent to 2,426 J. The analog output of the hygrometer was continuously monitored on a chart recorder.

The mice were singly exposed in the waveguide while housed in a circular Plexiglas cage (3.4 × 6.7 cm). Mineral

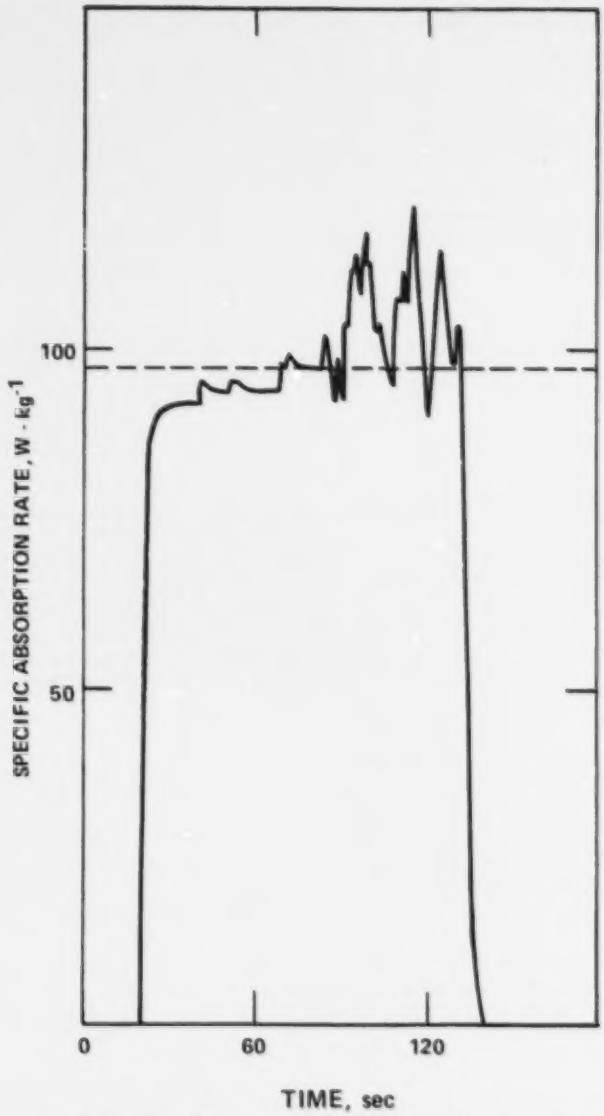

FIG. 2. Example of time course of power absorption by a mouse exposed to 2,450-MHz microwaves in waveguide-exposure system. *Dashed line* represents average specific absorption rate (SAR) for total time of microwave exposure. Area under SAR curve is equal to total absorbed heat from microwave source.

oil was placed beneath the cage to prevent moisture from urine and feces from entering into the water loss measurement.

Experimental protocol. The mice were kept inside the waveguide for approximately 60 min to allow equibration, indicated by a stable EWL of approximately 0.05 mg·g^{-1}·min^{-1}, to take place. The mice were exposed to an incident power of 3 or 6 W, which resulted in average SARs of 47.4 ± 0.8 and 93.4 ± 1.57 W·kg^{-1}, respectively. The average exposure time was 272 s at 3 W incident power and 150 s at 6 W forward power. The mice were used repeatedly over a 36-day period of experimentation. Approximately four to six exposures were made over a 3- to 5-h period. At least 48 h elapsed between experiments on the same animal.

Data analysis. The total amount of heat dissipated by evaporation that could be attributed to microwave exposure was taken as the integral of the rise-fall curve of EHL (W·kg^{-1}) over time. This integration (W·kg^{-1}·s^{-1}) yields total heat loss by evaporation per unit body weight dissipated in response to microwave exposure. It should be noted that the units of evaporated heat (J·g^{-1}) are also the same for the calculation of absorbed microwave energy (e.g., see Fig. 2). Significant interactions between absorbed and evaporated heat within and between the high- and low-SAR groups were assessed with a one-way analysis of variance employing a general linear model procedure (14).

The ratio of evaporated to absorbed heat yields a unitless number that describes the sensitivity of the mouse to microwave heat absorption. The higher the ratio the greater the amount of heat dissipated by evaporation per unit heat absorbed. To use terminology from control systems analysis (19), if the absorbed heat is treated as an error signal by the animal and the heat loss as a motor (output) response, then the motor output-to-error signal ratio is equal to the open-loop gain (OLG) of EHL system (10). This method of calculating OLG is similar to that used in past studies (22, 25), except that in the present study EHL replaces rectal temperature as the motor response and whole-body absorbed heat replaces hypothalamic temperature as the error signal.

Temperature measurements. In separate experiments deep body temperature was measured in pentobarbital sodium-anesthetized (~70 mg·kg^{-1}) mice during exposure to microwave radiation. A Vitek temperature probe (model 101), designed to measure temperatures in a microwave field, was inserted 18 mm past the anal sphinctor. It was necessary to anesthetize the animals because the waveguide construction prevents the use of conventional chronic temperature measurements in awake free-moving animals. The mice were give 30 s exposure to microwaves at high and low incident power while deep body temperature and SAR were continuously recorded.

RESULTS

Typical EHL responses of a mouse exposed to 2,450-MHz microwaves at a low and high SAR are presented in Fig. 3. The areas under the EHL and power-absorption curves represent the quantities of heat dissipated by evaporation and absorbed from the microwaves, respectively. The high and low SARs in Fig. 3 represent nearly identical heat loads (16.1 and 15.6 J·g^{-1}, respectively); however, the EHL response of the high-SAR exposure is 3.9 times greater than the low-SAR exposure.

Multiple regression analysis indicated a significant increase in EHL when mice were exposed at the high vs. low SAR ($F = 5.0$, $P < 0.02$) (Fig. 4). At 47 W·kg^{-1} the effect of absorbed microwave heat on evaporated heat was modeled with a linear equation where evaporated heat = −12.1 + 1.15 • absorbed heat; at 93 W·kg^{-1}, evaporated heat = −4.8 + 0.929 • absorbed heat. Thus at 47 W·kg^{-1} a heat load of 10.5 J·g^{-1} had to be exceeded

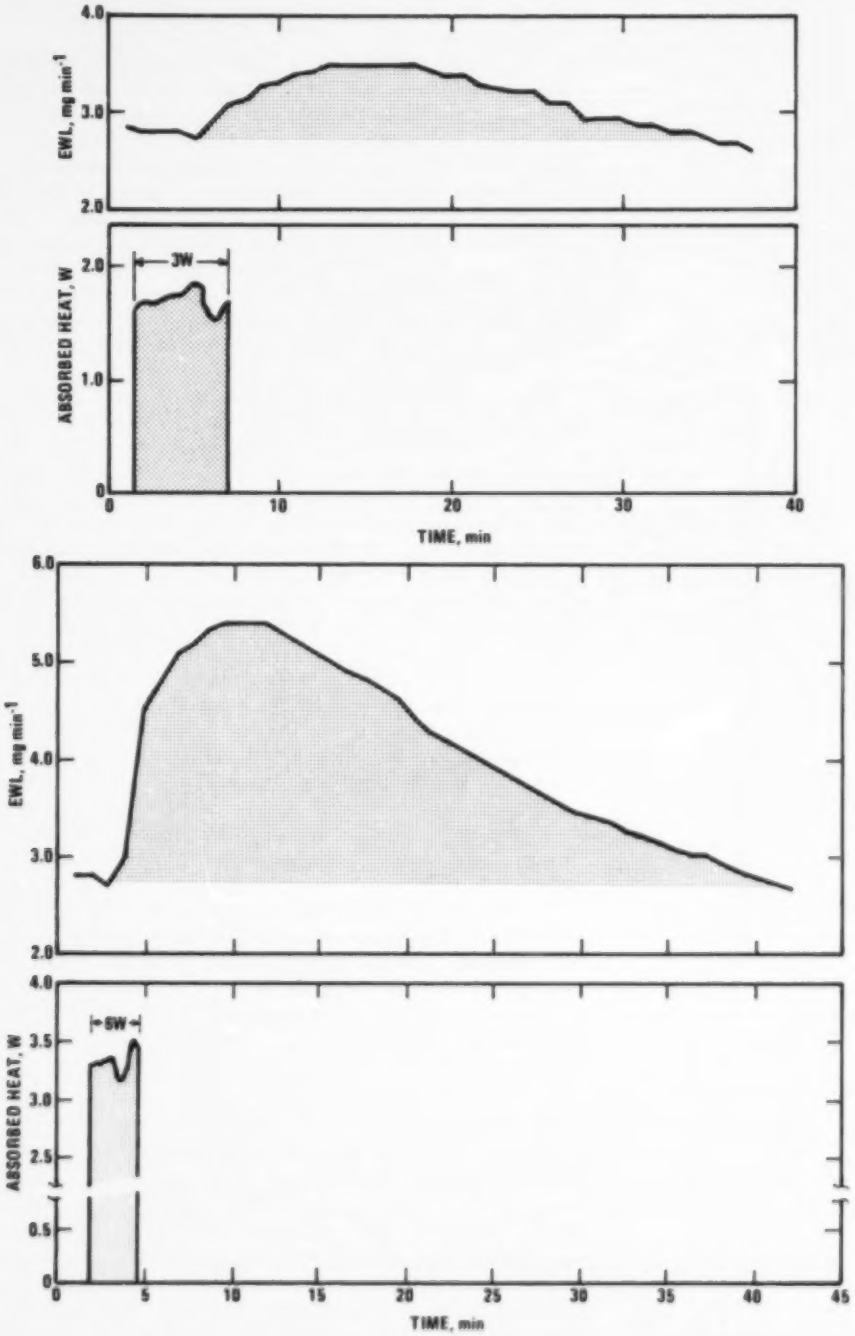

FIG. 3. Effect of microwave exposure at incident powers of 3 and 6 W on rate of whole-body evaporative water loss (EWL). *Shaded area* under rate of EWL (*upper curves*) is used to calculate total heat dissipated by evaporation as a result of heat load (*shaded area on lower curves*) absorbed from microwave exposure. Data were collected from 1 mouse on 1 day.

before EHL increased; at 93 $W \cdot kg^{-1}$ the threshold was reduced to 5.2 $J \cdot g^{-1}$. Above threshold the slopes of the regression lines for both SARs were statistically indistinct. Therefore, the difference in threshold was the major factor attributing to the significant elevation in EHL in the high-SAR group.

In this study the overall sensitivity of the EHL system is defined as the total heat dissipated by evaporation ($-J \cdot g^{-1}$) divided by the absorbed heat load ($J \cdot g^{-1}$). This dimensionless negative ratio, defined as OLG, would be zero at any heat load below threshold. OLG as a function of heat load above threshold can be calculated from the linear equations in Fig. 4 by dividing the equation by the absorbed heat load. For example, at 47 $W \cdot kg^{-1}$ Y/X (OLG) = $-12.1/X + 1.15$, where Y is evaporated heat and X is absorbed heat; for an SAR of 93 $W \cdot kg^{-1}$ OLG = $-4.8/X + 0.929$. Because these equations are hyperbolic functions, OLG initially rises abruptly with increas-

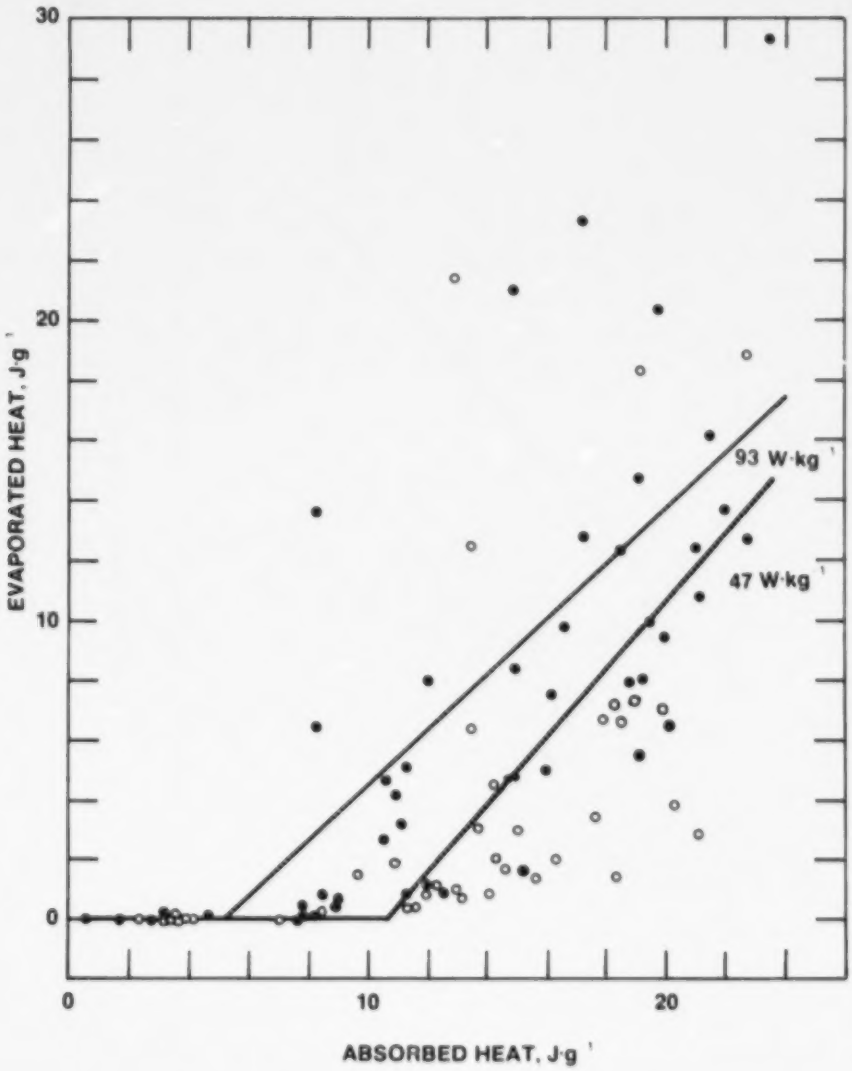

FIG. 4. Relationship between absorbed and dissipated heat in mice absorbing microwave energy at specific absorption rates (SAR) of 47 (*open circles*) and 93 $W \cdot kg^{-1}$ (*filled circles*). In both SAR groups there is a significant elevation in evaporated heat with increasing absorbed heat ($F = 20.3$, $P < 0.001$). Doubling SAR causes a large reduction in threshold and a slight decrease in slope of regression line ($F = 5.0$, $P < 0.02$). All data above threshold were plotted with a linear equation, $Y = a + bX$. At 47 $W \cdot kg^{-1}$, $a = -12.1 \pm 3.9$ (SE), $b = 1.15 \pm 0.24$, $r = 0.6368$; at 93 $W \cdot kg^{-1}$, $a = -4.8 \pm 4.1$, $b = 0.929 \pm 0.26$, $r = 0.4804$.

TABLE 1. *Open-loop gain as a function of absorbed microwave heat load in mice exposed to SARs of 47 or 93 $W \cdot kg^{-1}$*

Microwave Heat Load, $J \cdot g^{-1}$	Open-Loop Gain	
	Low SAR*	High SAR†
4		
8		0.32
12	0.14	0.52
16	0.39	0.62
20	0.54	0.68
24	0.64	0.72
28	0.71	0.75
32	0.77	0.77
36	0.81	0.79
40	0.84	0.80

SAR, specific absorption rate; OLG, open-loop gain; X, microwave heat load; $X_1 \geq 10.5$, $X_2 \geq 5.2$ $J \cdot g^{-1}$. Note tendency for OLG to asymptote as heat load increases beyond 32 $J \cdot g^{-1}$. * $OLG = -12.1/X_1 + 1.15$. † $OLG = -4.8/X_2 + 0.929$.

ing heat load and then gradually reaches an asymptote (Table 1). Because heat loads of less than 28 $J \cdot g^{-1}$ were used in the present study, OLG was significantly greater in the high-SAR group. It is unlikely that the animals could have tolerated whole-body heat loads much greater than 30 $J \cdot g^{-1}$.

There was a direct correlation between the rate of rise in deep body temperature and SAR (Fig. 5). The mean SARs at the incident powers of 3 and 6 W were found to be equivalent to increases in deep body temperature of 0.026 and 0.049°C/s, respectively.

DISCUSSION

A principal aspect of this study has been to demonstrate, not surprisingly, that mice thermoregulate. In the context of this report, thermoregulation implies that a given amount of heat is dissipated by EHL in response to a microwave heat load. The amount of heat loss

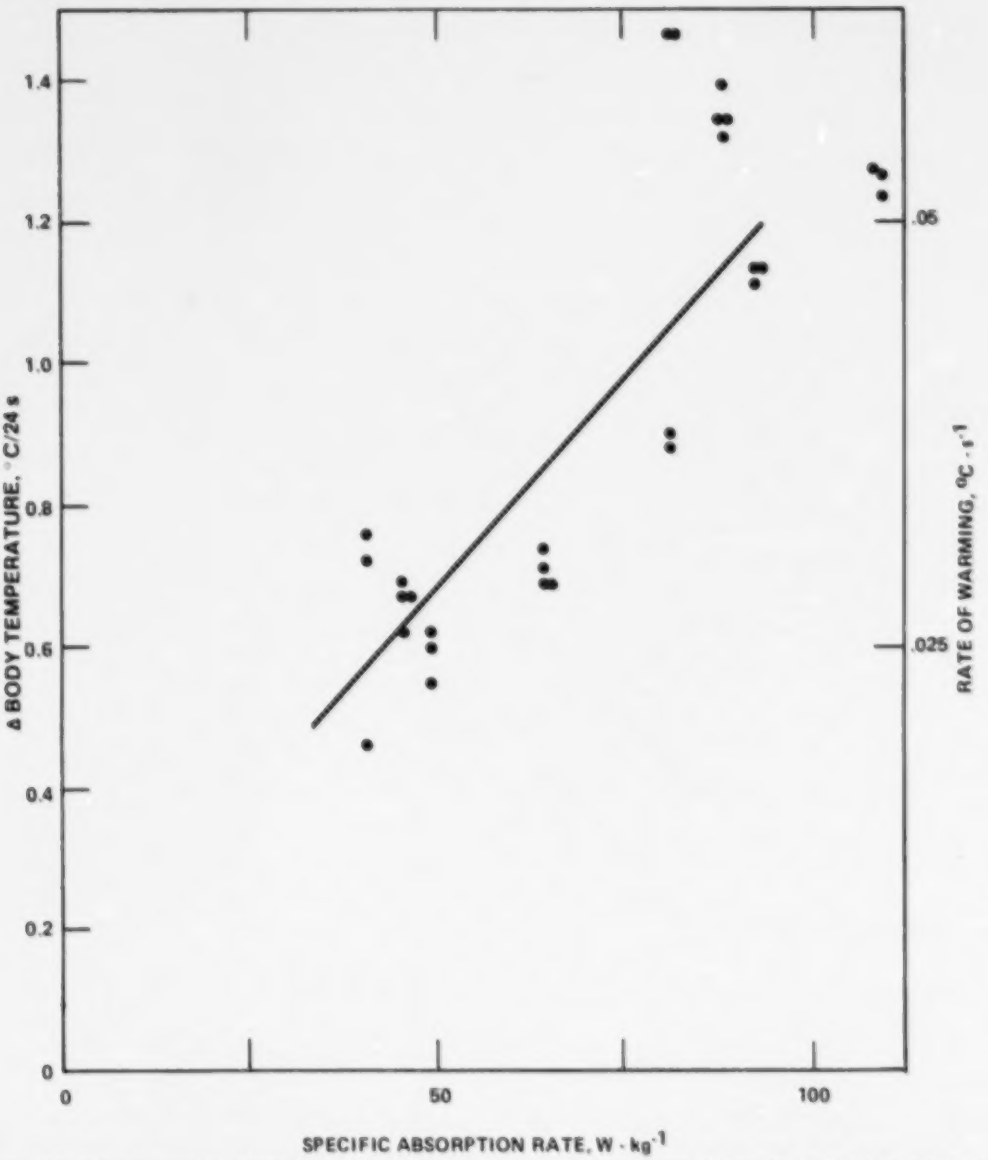

FIG. 5. Relationship between specific absorption rate and increase in colonic temperature of anesthetized mice exposed to 2,450-MHz microwaves.

through EHL can be calculated with the following equation

$$\text{heat loss, } J \cdot g^{-1} = \text{OLG (heat absorbed} - \text{threshold)}$$

Because the values of OLG and threshold are dependent on the rate of heat absorption, it is evident that the control of EHL involves an integrative (responds to total heat load) and differential capacity (responds to rate of heat absorption). Rate of heating implies that the temperature(s) of specific body parts will be elevated in a specified time, whereas an increase in total body heat simply refers to an increase in average body temperature without respect to time. Thus, because total heat loss by evaporation is controlled by the magnitude of OLG and threshold, which are in turn controlled by the rate of heat absorption, these data indicate that temperature regulation supersedes thermoregulation.

Rodents can dissipate heat by evaporation via water loss through respiration (9), spreading saliva on the fur (11), and insensible water loss through the skin (24). Rodents are not true panters but do undergo an increase in respiratory rate and respiratory water loss at high ambient temperatures (9). It will be of interest to show which of these three routes of EHL is most affected by the rate of heat absorption.

Previous studies have indicated that an animal the size

of a mouse would have an OLG less than 1.0, whereas in larger mammals such as the rabbit and dog OLG is equal to or greater than 10.0 (13). An OLG of 1.0 would be expected when EHL is the only route of heat loss (8). That is, the animal dissipates an amount of heat equal to what is absorbed. The intent of this study was not to measure the steady-state response of the controller, but instead, to assess the dynamic properties of a heat-dissipating motor output when a regulated system is given a transient thermal load.

The control of body temperature in mammals is thought to be based on a "set-point mechanism" whereby the controller attempts to maintain the central and peripheral temperatures within endogenously generated set-point temperatures (3, 12, 22). Alternate hypotheses have proposed that the control of body temperature is an indirect phenomenon resulting from the regulation of whole-body heat loss (17, 18, 26). Webb et al., (26) found that oxygen consumption during exercise was closely associated with whole-body heat loss and not correlated with rectal or mean skin temperature. Since an anatomical structure for the generation of a set-point temperature has not been located, it has been argued that temperature control is achieved by a balance between integrated cold and integrated warm inputs (24). Moreover, a model of temperature control in humans indicates that the control of deep body temperature is an indirect result of the control of heat storage (17, 18). The fact that many tissues and organs are thermal sensitive and, when heated, can effectively drive heat dissipation is strong support for a thermal-exchange model rather than a one- or two-temperature set-point model (24). The data in the present study do not necessarily support the set-point model of temperature control, but they strongly oppose the idea that the regulatory system is driven solely by absorbed heat per se. Instead, the data indicate that the heat-dissipating system is strongly affected by the rate of heat absorption.

This study differs from others since it distinguishes the importance between total heat exchange and actual tissue temperature on the activation and sensitivity of EHL. Elevating SAR not only increases the rate of temperature change but also increases the overall elevation in body temperature on the termination of microwave exposure. It remains to be seen whether the mouse responds to the overall change in temperature or to the rate of change.

Why is the heat-dissipating system of the mouse more sensitive to the rate of heat absorption than to total heat exchange? It is possible that by adjusting the OLG and threshold of EHL to the rate of heating the animal may be able to anticipate potentially large thermal loads. Anticipating thermal loads may be critical for the relatively slow-response nature of the temperature-regulatory system. For example, the time required to increase EHL in response to a heat load can take several minutes in humans (17, 18). In Fig. 3 the time difference between the onset of exposure to 6 W and the peak increment in EHL is approximately 7 min. If the temperature-regulatory system could maintain a second-to-second evaluation of total body heat and evaporate the heat in the same amount of time required for absorption, then a pure thermoregulatory system would be feasible. But since the activation of the heat-dissipating system is extremely slow in relation to the rate of heat absorption from the environment, it seems probable that the sensitivity of the system (i.e., the magnitude of OLG and threshold) must be, at the onset of heat exposure, adjusted to the rate of heat absorption to maintain a stable body temperature.

Stolwijk and Hardy (23) concluded that rate sensitivity was important in physiological control because it strongly opposed rapid departures of the controlled variable from the steady state. The fact that thermal receptors are highly sensitive to the rate of change in temperature (5, 7) further supports the hypothesis of a rate-controlled system. The control of sweating in humans is greatly dependent on the rate of change in skin temperature (20, 28).

The reciprocal of the slope of warming rate vs. SAR in Fig. 5 has the dimensions of specific heat and is equal to 2.03 $J \cdot g^{-1} \cdot °C^{-1}$. This value is considerably less than the specific heat of whole animals, which is approximately 3.47 $J \cdot g^{-1} \cdot °C^{-1}$. This discrepancy is due to the nonuniform manner in which animals absorb microwave radiation. At resonant microwave frequencies a greater proportion of the absorbed energy is deposited internally. If the absorbed energy was distributed evenly then the reciprocal of the heating curve would equal the specific heat of the whole animal. A steeper slope indicates that more energy is being deposited internally than peripherally.

Microwave radiation of sufficient intensity can affect numerous thermoregulatory motor outputs including metabolism (15), peripheral blood flow (1), and behavior (2). Radiation at 2,450 MHz resonates with a mass the size of mouse (6), which means that a large proportion of the absorbed microwave energy is deposited internally. This mode of heating is clearly not a normal form of heat stress as would occur during convective or radiant heating. However, applying heat with the microwave waveguide offers unique advantages over other types of heating because the amount and rate of heating can be quantitated. The capacity to directly calculate heat absorption may lead to a better understanding of body-temperature control.

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